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Letter to the editor

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*Dear Editor*

I read the article of Gilles-Eric Séralini et al. (2012, on-line version) entitled "Long term toxicity of a Roundup herbicide and a Roundup-tolerant genetically modified maize" with great interest. The importance of the topic and the relevant socio-economic implications drive me, due to my personal research experience on maize, to put forward an important issue that requires the careful consideration of the authors.

It is well known that maize kernels can be affected by fungal infections (mainly fungi belonging to the genus of *Fusarium* and *Aspergillus*) leading to the contamination of grains with mycotoxins such as aflatoxins and in particular fumonisins (Shephard et al., 1996; Pilu et al., 2011). These toxins are associated with a number of serious animal and human diseases, in particular cancer (Ross et al., 1992). For this reason the FAO/WHO Expert Committee on Food Additives, the U.S. Food and Drug Administration and the European Union have established for direct human consumption the threshold of fumonisin content in maize at the level of 1 ppm (Rule EU 1126/2007). It is also well known that incorrect conditions for seed and flour storage (e.g. high temperature and moisture) cause increases in the levels of contamination.

The authors wrote in Materials and Methods paragraph (2.2. Plants, diets and chemicals): "The varieties of maize used in this study were the R-tolerant NK603 (MonsantoCorp., USA), and its nearest isogenic non-transgenic control. These two types of maize were grown under similar normal conditions, in the same location, spaced at a sufficient distance to avoid cross-contamination." This distance is of about two hundred meters, which may determine in some cases dramatic differences in the kernel quality due to slight environmental differences in the field. This work does not report or mention the analysis of the mycotoxins content of the material obtained.

Hence my questions for the authors are:

(1) Did they assay the mycotoxins content in the seeds used to produce the feed?
(2) Did they also monitor the mycotoxin content in the feed during the entire time of the experiment?
(3) If the values of the mycotoxins content were different among the different diets, how did they normalize the data collected to take into consideration the carcinogenic effects of these toxins?

I think that the readers of this important journal should have these answers from the authors to better understand and evaluate the results obtained in this work.

## References

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